

Supporting Text

Our model estimates the mean number of deaths from an airborne anthrax attack under five different response strategies. The values of the model parameters are listed in Table 2. We use a Gaussian plume model (1) adapted for an instantaneous point release. The model is identical to the one used by Meselson *et al.* (2) for the Sverdlovsk outbreak, except for the values of the height h and size Q of the release, and hence assumes dispersion parameters given by Briggs (1) for neutral stability in open country, and ignores infectivity decay, deposition, protection from buildings, secondary aerosolization, and limits on vertical mixing. The number of spores inhaled by a person x meters directly downstream of the release point, and y meters crosswind is

$$s(x, y) = \frac{bQ}{\pi u \sigma_y \sigma_z} e^{-\frac{y^2}{2\sigma_y^2} - \frac{h^2}{2\sigma_z^2}} \quad [1]$$

where $\sigma_y = 0.08x/\sqrt{1 + 0.0001x}$, $\sigma_z = 0.06x/\sqrt{1 + 0.0015x}$, u is the wind speed, and b is the breathing rate. The height (100 m) can represent a release from the top of a tall building or from a low-flying aircraft. The release amount corresponds to ≈ 1 kg of anthrax (2).

In our numerical computations, we consider the region up to $X = 201$ km downwind and $\pm Y = 18$ km crosswind. Note that with a wind speed of $u = 5$ m/s and an early evening release, airborne anthrax spores would travel ≈ 200 km before being inactivated by ultraviolet rays from the sun; extending X from 201 to 1,001 km only increases the number of people infected in the base case by 4.0%. Although our model is formulated by using a continuous state space, in the computations we discretize (x, y) space by using a grid of 1 km to calculate the inhaled spore count. This region is divided into an urban area that is within

30 km downwind of the point of release and an outlying rural area. The population density $\theta(x, y)$ is taken to be $\theta_u = 10^4$ people per km^2 (3) in the urban region and $\theta_r = 10^2$ people per km^2 in the rural area, which generates 10.8 million urban residents and 0.7 million rural residents in our study region. The probability density function (pdf) of the population's age is $f(a)$, which is assumed to be uniformly distributed (with maximum age 85) within each of the following four age classes: 33.4% 0 – 24, 31.5% 25-44, 22.4% 45-64, and 12.7% 65-85 (4). People do not change location or age in our model.

Because of the strong age dependence in the dose-response relationship of inhalation anthrax (2, 5, 6), we incorporate age into the probit model used by Glassman (7), so that

$$P(s, a) = \Phi(\alpha + \beta \log s + \gamma a + \delta a^2) \quad [2]$$

is the likelihood that a person of age a gets infected from inhaling s spores (8), where $\Phi(\cdot)$ is the standard normal cumulative distribution function (cdf). The four parameters in Eq. 2 were estimated by a least-squares analysis using the ID_{50} and ID_{10} values, together with ages 15, 35, 55, and 75, in table 3 of ref. 9; see Fig. 2a.

Let $I_j(x, y, a, t)$ denote the density of people in disease stage j at location (x, y) , age a , and time t , where $j = 0, \dots, 4$, denotes uninfected, incubation, prodromal, fulminant, and death, respectively. Even though monkey studies reveal that the incubation period is inversely related to dose (10-12), we performed a statistical analysis of the data in ref. 2 and were unable to detect a statistically significant relationship between incubation period and either dose or age; a dose-dependent incubation period would increase the death toll in our model by exacerbating congestion at the hospital queues in the urban service zones. The duration of disease stages $j = 1, 2, 3$ have cdf $F_j(t)$, pdf $f_j(t)$, survival function $\bar{F}_j(t)$,

and hazard rate function $h_j(t) = f_j(t)/\bar{F}_j(t)$. These disease durations are taken to be log normal, where the log durations have mean r_j (i.e., the median disease period duration is $m_j = e^{r_j}$) and variance σ_j^2 . The parameters m_1 and σ_1^2 are taken from an analysis of the Sverdlovsk release (13). For $j = 2, 3$, the parameter σ_j^2 was chosen so that the dispersion factor $d_j = e^{\sigma_j^2} = \sqrt{2}$, and hence 95% of the disease period durations fall within half of the median and twice the median (13). The parameter r_j was chosen to coincide with a mean prodromal period of 2.5 days (in the absence of treatment) and a mean fulminant period of 1.5 days (5, 13).

The attack occurs at time 0, and intervention begins at time $\tau = 48$ h. At time τ , the aerosol would be fully dispersed (14). At the time of the attack, we have $I_1(x, y, a, 0) = \theta(x, y)f(a)P(s(x, y), a)$, and $I_0(x, y, a, 0) = \theta(x, y) - I_1(x, y, a, 0)$, and until intervention begins (i.e., for $t \in [0, \tau]$), the system state is given by

$$I_0(x, y, a, t) = I_0(x, y, a, 0), \quad [3]$$

$$I_1(x, y, a, t) = I_1(x, y, a, 0)\bar{F}_1(t), \quad [4]$$

$$I_2(x, y, a, t) = I_1(x, y, a, 0)[F_1(t) - \int_0^t f_1(u)F_2(t-u)du], \quad [5]$$

$$I_3(x, y, a, t) = I_1(x, y, a, 0)[\int_0^t f_1(u)F_2(t-u)du - \int_0^t \int_0^u f_1(v)f_2(u-v) dv F_3(t-u) du], \quad [6]$$

$$I_4(x, y, a, t) = I_1(x, y, a, 0)[\int_0^t \int_0^u f_1(v)f_2(u-v) dv F_3(t-u) du]. \quad [7]$$

To model intervention, we divide the 201×36 -km region into square service zones covering a 9-km² urban area of 9×10^4 people or an 81-km² rural area of 8,100 people. In New York City, a point of distribution for antibiotics serves ≈ 33 K people, and a “911” hospital serves ≈ 127 K people (Hauer, J. M., Congressional Testimony, Senate Health, Education,

Labor and Pensions Committee, March 25, 1999); our choice of 90 K people in an urban zone is between these two values. A two-stage (antibiotics and hospital) queueing system is located in each zone and provides service only to people in that zone. As discussed later, the local servers at these queues are assisted by a set of mobile hospital servers. Individuals continue their disease progression while waiting in queue.

Combination antibiotics have two effects in our model. First, they are assumed to be 90% effective ($e_1 = 0.9$) at preventing symptomatic disease when administered during the incubation period. While the antibiotics themselves are extremely effective (15), the 10% ineffectiveness is assumed to be primarily due to nonadherence of patients. Although full adherence was $< 50\%$ among postal workers in 2001 (16,17), adherence would likely be higher in a subsequent large-scale attack. Also, prodromal individuals who have received antibiotics do not progress to the fulminant stage with probability $e_2 = 0.4$. It has been claimed that 45-80% of severely symptomatic inhalational anthrax patients die if they do not receive immediate aggressive treatment in a state-of-the-art hospital (www.anthrax.mil). The efficacy of combination antibiotics administered during the febrile stage, coupled with aggressive supportive care (e.g., intravenous antibiotics, draining of pleural effusions), was unexpectedly high in the fall of 2001 (5). It is not clear whether this was due to the care or to the particular anthrax strain (10). With probability $1 - e_2$, these individuals have a prodrome duration (their prodromal clock is restarted if they receive antibiotics while in the prodromal stage) that is log normal with median $m_2 = 2$ days, dispersion factor $d_2 = \sqrt{2}$, and hazard rate function $h_2(t)$. People who complete their hospital care while in the prodromal stage survive with probability 1, but neither antibiotics nor hospital care are capable of preventing

death once someone enters disease stage 3 (5, 18). Note that while Sverdlovsk victims who had late onset also had a higher survival rate (2), this may be due to delayed administration of antibiotics and vaccines after the release.

In the five response policies in Table 1, a geographical ring, which grows over time, is used to specify which asymptomatics get in the antibiotics queue and at what time. All people who are symptomatic at time τ enter the antibiotics queue at time τ (the time intervention begins), and anyone who becomes symptomatic after time τ is placed in the queue at this later time if he did not previously join the queue (via the ring). A person enters the hospital queue as soon as he both develops symptoms and receives antibiotics, which can occur in either order. While antibiotics are administered only by local servers, hospital care is administered by both local servers and mobile servers, the former representing workers in a specific service zone, and the latter representing state and federal employees who can be dynamically allocated across the entire region. The density of local servers at queue i (where $i = A$ is the antibiotic queue and $i = H$ is the hospital queue) is $n_i(x, y)$, which we assume is proportional to the population density at each location. The density of antibiotic servers $n_A(x, y)$ was chosen so that, given the service rate μ_i (see below), antibiotics can be distributed to the entire population in 4 days. The number of emergency nurses in the United States in June 2001 was 89,300 and the number of emergency physicians in 2000 in the United States was 32,020 (www.aaem.org). Summing these two quantities, dividing by 286 million people and dividing by three shifts of workers per day gives the density n_H shown in Table 2. In addition, there are m_H mobile hospital servers who are used to handle the overflow of patients who cannot be served locally in a timely fashion; these servers are

available $\tau_m = 18$ h after the attack is detected (Hauer, J. M., Congressional Testimony, Senate Health, Education, Labor and Pensions Committee, March 25, 1999). We assume that each server at queue i is capable of serving patients at rate μ_i . Our mean service time for antibiotic distribution of 7 min in Table 2 is close to the implied mean service time of about 5.7 min if three shifts are used, and 8.5 min if two shifts are used (Hauer, J. M., Congressional Testimony, Senate Health, Education, Labor and Pensions Committee, March 25, 1999). The mean hospital care time of 6 h in Table 2 is intended to include several hours to stabilize a patient (e.g. provide intravenous antibiotics, monitor metabolites and electrolytes) and several additional hours to provide reactive care (e.g., rehydration, thoracentesis, chest tube placement) (5).

For notational purposes, we need to distinguish between the prodromal patients in the hospital queue who may (with probability $1 - e_2$), or may not, progress to the fulminant disease stage. To this end, we define disease stages $\tilde{2}$ and $\hat{2}$ for people in the hospital queue who have finite and infinite prodromal durations, respectively. For $i = A$, $j = 0, 1, 2, 3$, and for $i = H$, $j = \tilde{2}, \hat{2}, 3$, define $Q_j^i(x, y, a, w, t)$ to be the density of people in queue i at time t who are in disease stage j at location (x, y) and have been in this disease stage for exactly w time units. Let $Q_j^i(x, y, a, t) = \int_0^t Q_j^i(x, y, a, w, t) dw$ be the density of people in queue i at time t of age a and in disease stage j at location (x, y) . Note that for $j = 0, 1, \hat{2}$, we need not keep track of people's "disease age" because people in stages 0 and $\hat{2}$ do not progress and the disease age of individuals in stage 1 equals t , since they were infected at time 0. In addition, let $U_1(x, y, a, t)$ be the density of people of age a in disease stage 1 at location (x, y) who have unsuccessfully received antibiotics and will progress to symptoms.

In the equations below, $A_j(x, y, a, t)$, $j = 0, 1$ are the arrival rates of asymptomatics to the antibiotics queue due to the ring strategy, and $S_j^i(x, y, a, t)$ are the service rates from queue i ; both are described in detail later. All transitions are due to these arrivals and services, or to disease progression. By our earlier discussion, the system state at time τ is $Q_2^A(x, y, a, w, \tau) = I_1(x, y, a, 0)f_1(\tau-w)\bar{F}_2(w)$, $Q_3^A(x, y, a, w, \tau) = I_1(x, y, a, 0) \int_0^{\tau-w} f_1(u)f_2(\tau-w-u) du \bar{F}_3(w)$, and $U_1(x, y, a, \tau) = Q_0^A(x, y, a, \tau) = Q_1^A(x, y, a, \tau) = Q_j^H(x, y, a, w, t) = 0$. In addition, $I_j(x, y, a, t) = 0$ for $j = 2, 3$, $t > \tau$. The dynamics for $t \geq \tau$ are given by

$$\frac{dI_0(x, y, a, t)}{dt} = -A_0(x, y, a, t), \quad [8]$$

$$\frac{dQ_0^A(x, y, a, t)}{dt} = A_0(x, y, a, t) - S_0^A(x, y, a, t)Q_0^A(x, y, a, t), \quad [9]$$

$$\frac{dI_1(x, y, a, t)}{dt} = -A_1(x, y, a, t) - h_1(t)I_1(x, y, a, t), \quad [10]$$

$$\frac{dQ_1^A(x, y, a, t)}{dt} = A_1(x, y, a, t) - [h_1(t) + S_1^A(x, y, a, t)]Q_1^A(x, y, a, t), \quad [11]$$

$$\frac{dU_1(x, y, a, t)}{dt} = (1 - e_1)S_1^A(x, y, a, t)Q_1^A(x, y, a, t) - h_1(t)U_1(x, y, a, t), \quad [12]$$

$$Q_2^A(x, y, a, 0, t) = h_1(t)I_1(x, y, a, t) + h_1(t)Q_1^A(x, y, a, t), \quad [13]$$

$$\frac{\partial Q_2^A(x, y, a, w, t)}{\partial w} + \frac{\partial Q_2^A(x, y, a, w, t)}{\partial t} = -[h_2(w) + S_2^A(x, y, a, t)]Q_2^A(x, y, a, w, t), \quad [14]$$

$$Q_3^A(x, y, a, 0, t) = \int_0^t h_2(w)Q_2^A(x, y, a, w, t) dw, \quad [15]$$

$$\frac{\partial Q_3^A(x, y, a, w, t)}{\partial w} + \frac{\partial Q_3^A(x, y, a, w, t)}{\partial t} = -[h_3(w) + S_3^A(x, y, a, t)]Q_3^A(x, y, a, w, t), \quad [16]$$

$$Q_2^H(x, y, a, 0, t) = (1 - e_2) \left[h_1(t)U_1(x, y, a, t) + \int_0^t S_2^A(x, y, a, t)Q_2^A(x, y, a, w, t) dw \right], \quad [17]$$

$$\frac{\partial Q_2^H(x, y, a, w, t)}{\partial w} + \frac{\partial Q_2^H(x, y, a, w, t)}{\partial t} = -[h_2(w) + S_2^H(x, y, a, t)]Q_2^H(x, y, a, w, t), \quad [18]$$

$$\begin{aligned} \frac{dQ_2^H(x, y, a, t)}{dt} = e_2 \left[h_1(t) U_1(x, y, a, t) + \int_0^t S_2^A(x, y, a, t) Q_2^A(x, y, a, w, t) dw \right. \\ \left. - S_2^H(x, y, a, t) \right] Q_2^H(x, y, a, t), \end{aligned} \quad [19]$$

$$Q_3^H(x, y, a, 0, t) = \int_0^t h_2(w) Q_2^H(x, y, a, w, t) dw, \quad [20]$$

$$\begin{aligned} \frac{\partial Q_3^H(x, y, a, w, t)}{\partial w} + \frac{\partial Q_3^H(x, y, a, w, t)}{\partial t} = S_3^A(x, y, a, t) Q_3^A(x, y, a, w, t) \\ - [h_3(w) + S_3^H(x, y, a, t)] Q_3^H(x, y, a, w, t), \end{aligned} \quad [21]$$

$$\begin{aligned} \frac{dI_4(x, y, a, t)}{dt} = \int_0^t \left[h_3(w) \left(Q_3^A(x, y, a, w, t) + Q_3^H(x, y, a, w, t) \right) + S_3^H(x, y, a, t) Q_3^H(x, y, a, w, t) \right] dw, \\ [22] \end{aligned}$$

and the total dead is $\int_0^\infty \int_0^X \int_{-Y}^Y I_4(x, y, a, \infty) dy dx da$.

It remains to specify the rates $A_j(x, y, a, t)$ and $S_j^i(x, y, a, t)$ appearing in the above equations for the various policies. The ring-based strategy behind $A_j(x, y, a, t)$ tracks the cumulative number of symptomatic anthrax cases per capita at location (x, y) by time t , assuming that this location has not entered the ring by time t . This quantity, which we call the observed anthrax burden, is $\int_0^\infty I_1(x, y, a, 0) da F_1(t)/\theta(x, y)$. The ring at time t consists of all locations that have burdens at least as large as the threshold p . Hence, location (x, y) enters the ring at time

$$t^*(x, y) = F_1^{-1} \left(\frac{p\theta(x, y)}{\int_0^\infty I_1(x, y, a, 0) da} \right) \quad \text{if} \quad \int_0^\infty I_1(x, y, a, 0) da > p\theta(x, y), \quad [23]$$

and $t^*(x, y) = \infty$ otherwise, where $F_1^{-1}(\cdot)$ denotes the inverse cdf of the incubation period. Because intervention does not begin until time τ , if we let $I_{\{x\}}$ denote the indicator function of the event x , then

$$A_0(x, y, a, \tau) = I_0(x, y, a, 0) I_{\{\tau \geq t^*(x, y)\}}, \quad [24]$$

$$A_1(x, y, a, \tau) = I_1(x, y, a, 0) \bar{F}_1(\tau) I_{\{\tau \geq t^*(x, y)\}}, \quad [25]$$

and, for $t > \tau$,

$$A_0(x, y, a, t) = I_0(x, y, a, 0) I_{\{t=t^*(x, y)\}}, \quad [26]$$

$$A_1(x, y, a, t) = I_1(x, y, a, 0) \bar{F}_1(t^*(x, y)) I_{\{t=t^*(x, y)\}}. \quad [27]$$

Finally, we specify the service rate terms $S_j^i(x, y, a, t)$. Define $Q_j^i(x, y, t) = \int_0^\infty Q_j^i(x, y, a, t) da$ to be the total density of people in disease stage j at location (x, y) in queue i at time t , $Q_2^H(x, y, t) = \sum_{j=\{\hat{2}, \hat{2}\}} Q_j^H(x, y, t)$ to be the total density of prodromals at the hospital queue at location (x, y) at time t , and $Q^i(x, y, t) = \sum_{j=0}^3 Q_j^i(x, y, t)$ to be the total density of people at location (x, y) in queue i at time t . The mass service policy is defined by

$$\begin{aligned} S_j^i(x, y, a, t) = & \mu_i \min\left(1, \frac{n_i(x, y)}{Q^i(x, y, t)}\right) \\ & + \frac{\mu_i}{Q^i(x, y, t)} (Q^i(x, y, t) - n_i(x, y))^+ \min\left(1, \frac{m_i}{\int_0^X \int_{-Y}^Y (Q^i(x, y, t) - n_i(x, y))^+ dy dx}\right) I_{\{t \geq \tau + \tau_m\}} \end{aligned} \quad [28]$$

for $i = A$, $j = 0, \dots, 3$ and for $i = H$, $j = \hat{2}, \hat{2}, 3$, where, for ease of presentation, we define $m_A = 0$. The first term on the right side of Eq. 28 represents service by the local servers. Note that the maximum departure rate per capita is $n_i(x, y)\mu_i$ and if there are more people in queue than servers, the departure rates are proportional to the relative densities of people in queue. The second term (for $i = H$) in Eq. 28 depicts the m_H mobile servers processing overflow people [i.e., leftover people in regions (x, y) where $Q^i(x, y, t) > n_i(x, y)$] in proportion to their overflow densities in queue. The symptomatic priority policy, where asymptomatic people in the antibiotics queue are only served if the number of servers exceeds

the number of symptomatics in queue, is defined by Eq. 28 for $i = H, j = \tilde{2}, \hat{2}, 3$, and by

$$S_j^A(x, y, a, t) = \mu_A \min\left(1, \frac{[n_A(x, y) - Q_2^A(x, y, t) - Q_3^A(x, y, t)]^+}{Q_0^A(x, y, t) + Q_1^A(x, y, t)}\right) \text{ for } j = 0, 1, \quad [29]$$

$$S_j^A(x, y, a, t) = \mu_A \min\left(1, \frac{n_A(x, y)}{Q_2^A(x, y, t) + Q_3^A(x, y, t)}\right) \text{ for } j = 2, 3. \quad [30]$$

The symptomatic/prodromal policy, which builds on the symptomatic policy by giving priority to prodromal patients over fulminant patients in the hospital queue (i.e., prodromals are served locally; then excess prodromals are served by mobile servers; then spare local servers serve fulminants; and finally spare mobile servers serve fulminants), is defined by Eqs. 29 and 30 and

$$S_j^H(x, y, a, t) = \mu_H \min\left(1, \frac{n_H(x, y)}{Q_2^H(x, y, t)}\right) + \frac{\mu_H}{Q_2^H(x, y, t)} [Q_2^H(x, y, t) - n_H(x, y)]^+ \min\left(1, \frac{m_H}{\int_0^X \int_{-Y}^Y [Q_2^H(x, y, t) - n_H(x, y)]^+ dy dx}\right) I_{\{t \geq \tau + \tau_m\}} \quad [31]$$

for $j = \tilde{2}, \hat{2}$, and

$$S_3^H(x, y, a, t) = \mu_H \min\left(1, \frac{[n_H(x, y) - Q_2^H(x, y, t)]^+}{Q_3^H(x, y, t)}\right) + \frac{\mu_H}{Q_3^H(x, y, t)} [Q_3^H(x, y, t) - (n_H(x, y) - Q_2^H(x, y, t))^+]^+ \min\left(1, \frac{[m_H - \int_0^X \int_{-Y}^Y (Q_2^H(x, y, t) - n_H(x, y))^+ dy dx]^+}{\int_0^X \int_{-Y}^Y [Q_3^H(x, y, t) - (n_H(x, y) - Q_2^H(x, y, t))^+]^+ dy dx}\right) I_{\{t \geq \tau + \tau_m\}}, \quad [32]$$

where the first term on the right side of Eq. 32 represents service of fulminant patients by spare (i.e., not serving prodromals) local servers and the second term is due to processing by spare mobile servers. The symptomatic-age policy and the symptomatic-age/prodromal

policy incorporate on top of the symptomatic and symptomatic/prodromal policies, respectively, a second layer of age-based priority at the antibiotics queue. Because this priority layer is conceptually similar to the disease-based priority at the antibiotics queue, we omit the detailed specifications of the symptomatic-age and symptomatic-age/prodromal policies.

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